Effect of Host Genotype Unit Area on Epidemic Development of Crown Rust Following Focal and General Inoculations of Mixtures of Immune and Susceptible Oat Plants

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ABSTRACT

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The effect of host genotype unit area (the ground area occupied by an individual, independent unit of the host population that is genetically homogeneous) on the effectiveness of oat mixtures in reducing epidemic development of crown rust was studied in two types of epidemics. The initial inoculum was confined to a single focus in the first, and the same amount of initial inoculum was distributed uniformly in the second. Plots with 100% pure-line susceptible plants were compared with those having two plant population mixtures, each consisting of 25% susceptible and 75% immune plants. The positions of the two genotypes were completely random in one mixture. In the other mixture, host genotype unit area was increased by aggregating 200 seeds of like genotype into randomly positioned blocks within plots. When the plots were artificially inoculated in a single focus in 1984, both mixtures reduced epidemic development relative to the pure-line susceptible check and there was a relatively small difference in the effectiveness of the two mixtures. When the same amount of initial inoculum was distributed uniformly over the plots in 1984, the completely random mixture greatly reduced rust development relative to the pure-line check, but the mixture of large genotype units did not. A similar result was obtained in 1983 when a natural spore shower resulted in an apparently random initial distribution of disease in the plots.

Additional key words: *Avena sativa*, epidemiology, multilines, *Puccinia coronata*.

The use of single, race-specific genes for resistance to plant pathogens often provides inadequate disease control because virulent genotypes are rapidly selected from the pathogen population (4,5). An alternative is to grow mixtures of plants that possess different race-specific resistance genes. The mechanisms contributing to disease control in such mixtures have been discussed (4,7,8,16).

The effectiveness of host mixtures has been demonstrated for foliar diseases of small grains (4,11,16,24) in which host plants are small and there is much opportunity for inoculum exchange among host genotypes. Vanderplank (22, page 144) hypothesized, however, that host mixtures may be less effective for crops with large plants for which a larger proportion of inoculum may be retained on the same genotype on which it was produced. A similar argument can be made in comparing random mixtures of plants (as in a multiline or cultivar mixture) with intercropping or interfield diversification.

Few studies have been conducted to determine the effects of plant size and host genotype aggregation on the effectiveness of host mixtures for the control of foliar plant pathogens. Barrett and Wolfe (1) changed the size of plants in a mixture of three barley (*Hordeum vulgare* L.) cultivars by taking advantage of an interaction between tillering ability and sowing density. Their results suggest that the effectiveness of the mixture in controlling powdery mildew (induced by *Erysiphe graminis* DC. f. sp. *hordei* Marchal) decreased with increasing plant size. Mundt and Browning (15) altered the spatial arrangement of near-isogenic oat (*Avena sativa* L.) lines to attain host mixtures with differing genotype unit areas. They defined genotype unit area as "the ground area occupied by an individual, independent unit of the host population that is genetically homogeneous." Mundt and Browning (15) found that all mixtures reduced epidemic development of crown rust (induced by *Puccinia coronata* Cda. var. *avenae* Fraser and Ledingham) and that increasing genotype unit area from 0.003 to 0.84 m^2 had no significant effect on the efficacy of the mixtures.

The epidemics in Mundt and Browning's (15) study were initiated with one disease focus per plot. A focus has been defined (3) as "the site of local concentrations of diseased plants or disease lesions, either about a primary source of infection or coinciding with an area favorable to establishment, and tending to influence the pattern of further transmission of the disease." Focal epidemics tend to occur when initial inoculum is low (26, page 151) and are observed in nature, e.g., with wheat (*Triticum aestivum* L.) stripe rust (induced by *Puccinia striiformis* West) (25) and with potato (*Solanum tuberosum* L.) late blight [induced by *Phytophthora infestans* (Mont.) d By.] (23). General epidemics, on the other hand, occur when the amount of initial inoculum is high and well dispersed (26, page 151). Roelfs and Martell (18) noted that, in the United States, foci of cereal rusts occur on winter grains where the pathogens overwinter, but that initial disease is "nearly randomly dispersed" on spring-planted cereals.

The purpose of our research was to determine if the effect of genotype unit area on the effectiveness of host mixtures for disease control depends on whether initial disease occurs in a single focus or is distributed more uniformly over the host population.

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MATERIALS AND METHODS

Experiment in 1983. Field plots at the Central Crops Research Station, Clayton, NC, were arranged in a 6 × 6 Latin square. Each plot was 6.1 × 6.1 m with 3.0 m between adjacent plots. Fertilizer was applied as necessary to maintain adequate growth of the crop.

Each plot was divided into 64 grids by using concrete reinforcing wire cut into 0.76 × 0.76 m sections. The plots were prepared on 16 March 1983 by raking back soil in each grid, scattering 200 seeds over the grid, and covering the seeds by raking soil back onto the plot. The germination percentage for seeds of both genotypes was ~95% or greater. Prior to planting, seeds were treated with carboxin (75W) at the rate of 0.94 g a.i/kg of seeds. The areas between plots and an area at least 3 m around the perimeter of the experiment were planted to Iowa Multiline E77 with a tractor-drawn grain drill.

The experiment consisted of a factorial arrangement of three oat populations and two methods of inoculation. The first oat population was a pure stand of C649 (C.I. 7555, the recurrent parent of the Iowa M-series multilines [10]). The other two populations each consisted of 25% C649 and 75% X421 (a near-isogenic line of C649 that carries an additional race-specific gene for resistance to *P. coronata*). In one mixture, 50 seeds of C649 and 150 seeds of X421 were planted in each 0.76 × 0.76-m grid. The seeds were mixed before planting. In the other mixture, all seeds within a grid were of the same genotype, with 16 of the grids planted to C649 and the other 48 planted to X421. The positions of genotype units within plots were completely random, except for the center four grids in plots of large genotype units. The center four grids in these plots were randomized separately so that one grid of C649 and three grids of X421 would be planted at the center of each plot.

We had planned to initiate one focal and one general epidemic in each replication for each of the three oat populations. Several inoculations with *P. coronata* race 264B were conducted between 6 and 21 May 1983. C649 is susceptible and X421 is immune to race 264B. The crown rust race was obtained from M. D. Simons and L. J. Michel, Iowa State University, in 1983 and was maintained on C649 oats in the greenhouse. Plants were inoculated by suspending uredospores in Isopar M oil and applying the suspension with a gas-propellent-type, thin-layer chromatography sprayer. For focal inoculations, the suspension was applied to the contiguous corners of the four central planting grids of each plot. For general inoculations, the same amount of initial inoculum was distributed uniformly over the entire plot. The artificial inoculations were largely unnsuccessful, and the epidemics were apparently initiated from an external source of inoculum that was virulent on C649 but not on X421 (see Results section).

Disease progression was quantified by sampling culms over time and counting the number of pustules per culm. Pustule counts were conducted on 28–29 May, 4–5, and 9–10 June. For the pure-line susceptible and completely random mixture treatments, 16 grids were sampled in an X-pattern in each plot. For the mixture of large genotype units, culms were sampled only in the 16 grids of C649. In the first pustule count, three culms per grid were counted in the pure-line susceptible plots and in the mixture of large genotype units. Six culms per grid were counted in the completely random mixture. In the two subsequent counts, one culm per grid was sampled in the pure-line susceptible plots and in the mixture of large genotype units. Four culms per grid were sampled in the completely random mixture so that, on the average, the same number of susceptible culms would be sampled for each treatment. We assumed that one-fourth of the culms sampled in the completely random mixture were C649 (the susceptible genotype). All counts were nondestructive. Data were expressed as the mean number of pustules per susceptible culm for each plot and each date.

Pustule counts for the susceptible genotype were plotted versus time to provide disease progress curves. The area under the disease progress curve (ADPC) was calculated for each plot as described by Shaner and Finney (21), except that the number of pustules per culm was substituted for the disease severity rating.

The probability levels for treatment differences were determined by analysis of variance using PROC GLM of the Statistical Analysis System (19). The $\log_{10}$ of ADPC was used in the analyses to better satisfy the assumption of homogeneity of variance. The experiment was analyzed as a factorial. The sum of squares for the main effect of oat populations was partitioned into two single-degree-of-freedom linear contrasts: completely random mixture versus mixture of large genotype units, and pure-line susceptible versus mixture of large genotype units.

Experiment in 1984. The experiment was located at the Peanut Belt Research Station, Lewiston, NC. The experimental design was the same as in 1983, except that adjacent plots were 6.1 m apart. Standard fertilization and weed control practices were used to maintain adequate growth of the crop.

Plots were prepared and seeds were planted in the same manner as in the 1983 experiment except that the sowing rate was adjusted to provide 200 viable seeds per planting grid. Prior to planting, seeds were treated with carboxin (75W) at 0.71 g a.i./kg of seeds.

Plants were inoculated on 5 May with the same isolate of *P. coronata* race 264B that was used the previous year. The isolate had been maintained on C649 and Markton oats in the greenhouse. Plants were injected below the uppermost node with a suspension of ~1.0 mg of viable uredospores per milliliter of distilled water to which Tween-20 was added at two drops per 100 ml. The suspension was injected until it exuded from the whorl or until 0.5 ml was administered. In the focally inoculated plots, 128 culms (32 in each of the contiguous corners of the four cental planting grids) were inoculated. In the generally inoculated plots, 128 culms (two in each of the 64 planting grids) were inoculated.

Disease progression was quantified by counting or estimating the number of pustules per susceptible culm. The first disease assessment was conducted on 24–25 May, before secondary spread had occurred. With the generally inoculated plots, all grids were searched and the total number of observed pustules was counted. In the plots inoculated focally, the total number of pustules was counted only in the inoculated grids at the center of each plot. Pustules were not observed in the uninoculated grids. The number of culms was counted in several representative grids in the experiment so that data could be expressed as the mean number of pustules per culm.

Culms were sampled in 16 grids of each plot in two subsequent assessments conducted on 5-6 and 13-14 June. With the mixture of large genotype units, two culms were selected from each of the 16 grids of C649 in each plot. With the pure-line susceptible plots and the completely random mixture, one grid was selected from each of 16 groups of four contiguous grids in each plot. Two culms per selected grid were assessed in the pure-line susceptible plots. For the 5–6 June assessment, eight culms per selected grid were assessed in the completely random mixtures so that, on the average, the same number of susceptible culms would be sampled for all treatments. For the last assessment (13–14 June), two rusted culms were selected from each of the 16 sampled grids in the completely random mixtures. A significant number of culms had senesced by the time the last assessment was made; so, for all treatments, only green culms were assessed on this date. For all three assessments, we assumed that 25% of the culms in the completely random mixtures were C649. All assessments, except the last, were nondestructive. For all treatments and all assessment dates, data were expressed as the mean number of pustules per susceptible culm per plot.

The ADPC was calculated for each plot as described for the 1983 experiment and was used to make treatment comparisons. Data from three columns of the Latin square were eliminated from the data set because these plots were severely damaged by herbicide carryover. Therefore, the experiment was analyzed as a randomized complete block design with three replications per treatment. The factorial arrangement of treatments was ignored and the sum of squares for treatments was partitioned into five single-degree-of-freedom linear contrasts because we were more interested in comparing oat populations within inoculation methods than with the main effects and interaction between oat populations and methods of inoculation. The five contrasts were:

pure-line susceptible (focal inoculation) versus mixture of large genotype units (focal), completely random mixture (focal) versus mixture of large genotype units (focal), pure-line susceptible (general inoculation) versus mixture of large genotype units (general), completely random mixture (general) versus mixture of large genotype units (general), and pure-line susceptible (focal) versus pure-line susceptible (general). All data were analyzed with PROC GLM of the Statistical Analysis System (19).

RESULTS

Experiment in 1983. The viability of inoculum used in the artificial inoculations was low and microclimate conditions were not always favorable for infection. Few or no pustules were observed at sites where inoculum was applied in a single focus, and focal development of disease was not observed in the plots at any time during the growing season. Oat crown rust did develop in all plots, but the initial disease seemed to be restricted to C649 plants and randomly distributed within plots with no obvious difference in the spatial pattern of disease in the focally versus generally inoculated plots. During the time plants were being artificially inoculated, there was heavy crown rust on winter oats in a breeding nursery less than 0.5 km from our experiment. Therefore, epidemics were most likely initiated from natural spore shower(s).

As expected, there was little difference in epidemic development between treatments receiving the focal as compared with the general artificial inoculations and, therefore, disease progress curves for each oat population were combined over both methods of inoculation (Fig. 1). The mean ADPC for the focal and general inoculations averaged over all three types of oat populations was 379 and 349 pustules-days, respectively. Probability levels for the main effect of inoculation method and the oat population × inoculation method interaction were 0.40 and 0.34, respectively.

The completely random mixture greatly reduced epidemic development on the susceptible genotype relative to the pure-line susceptible check (Fig. 1). The mixture of large genotype units was much less effective, however, and the disease progress curve for this treatment was more similar to the pure-line check than to the other mixture treatment (Fig. 1). Averaged over both inoculation methods, the ADPC was 567, 393, and 133 pustules-days for the pure-line check, the mixture of large genotype units, and the completely random mixture, respectively. The probability level for the difference in the ADPC between the two mixture treatments was <0.0001. The probability level for the difference in the ADPC between the pure-line susceptible check and the mixture of large genotype units was 0.07.

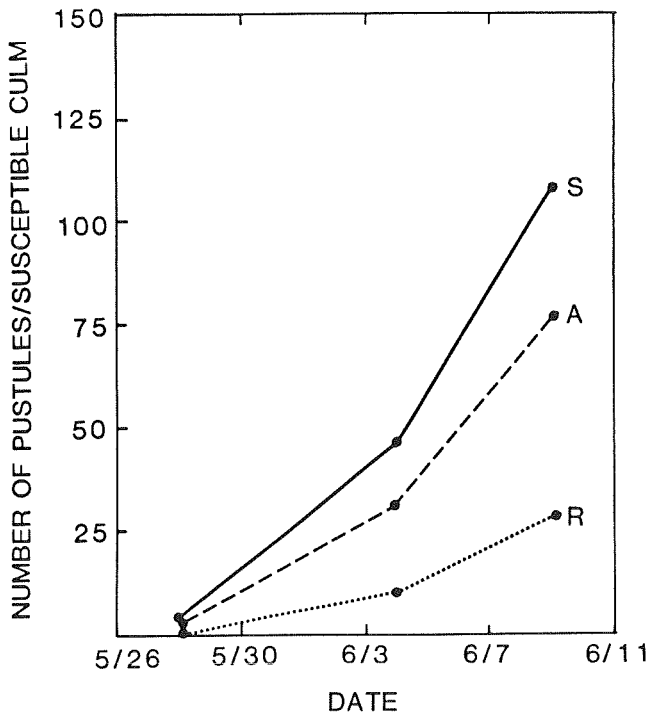

Fig. 1. Oat crown rust epidemics initiated in 1983 from a natural source of inoculum that resulted in an apparently random distribution of initial disease in the plots. Number of pustules on susceptible (C649) oat culms in field plots. Curve S, disease progress curve for a pure stand of C649. Curve A, disease progress curve for a mixture of 25% C649 and 75% X421 (immune) in which seeds were aggregated into blocks of 200 seeds of like genotype. Curve R, disease progress curve for a completely random mixture of 25% C649 and 75% X421. Each point is the mean of 12 replications.

Experiment in 1984. There was a definite difference in the spatial pattern of disease in the focal versus general treatments in 1984. A distinct central focus of rust developed in the focally inoculated, but not in the generally inoculated plots. Observations indicated that the inoculum was virulent on C649 but not on X421. No evidence of infection from natural sources of inoculum was found in C649 check plots in an adjacent oat experiment examined on 4 June.

Some of the artificial inoculations were unsuccessful, so initial disease did not develop in all portions of the generally inoculated plots. Of the 16 groups of four contiguous grids observed in each of the three pure-line susceptible plots on 24–25 May, pustules were found in 15, 11, and 16 of the groups. The corresponding values for the completely random mixture were 5, 8, and 5, and for the mixture of large genotype units 6 (of a possible 10), 7 (of a possible 12), and 7 (of a possible 11). There were fewer infected groups of plants in the mixtures because three-fourths of the inoculated plants were immune. There were fewer than 16 groups of four grids that could have been infected in the mixtures of large genotype units because some contiguous groups of four grids contained more than one susceptible grid, while others contained none. In addition, it is very likely that some infected plants were missed during searches through the grids. Averaged over all three oat populations, 30% fewer pustules were found in the generally inoculated plots than in the focally inoculated plots in which inoculated plants were aggregated and, therefore, easier to find.

With focal epidemics, both mixtures reduced epidemic development relative to the pure-line susceptible populations (Fig. 2). The ADPC for the pure-line check, the mixture of large

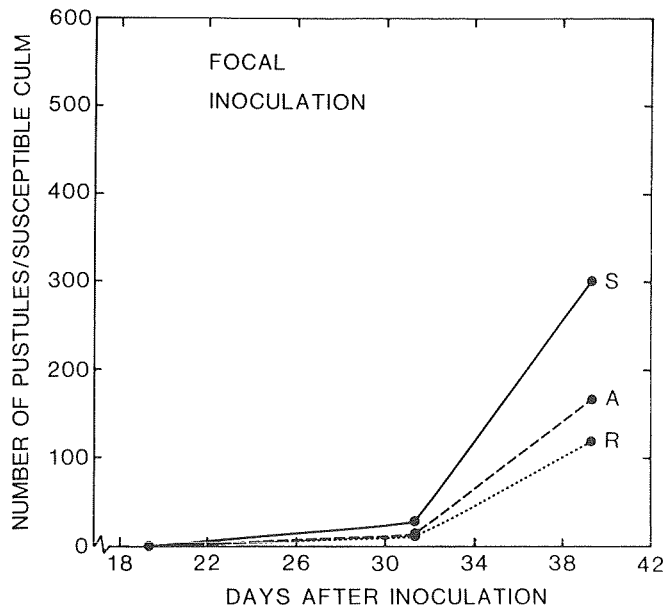

Fig. 2. Oat crown rust epidemics initiated in 1984 by artificial inoculation at a central focus in each plot. Number of pustules on susceptible (C649) oat culms in field plots. Curve S, disease progress curve for a pure stand of C649. Curve A, disease progress curve for a mixture of 25% C649 and 75% X421 (immune) in which seeds were aggregated into blocks of 200 seeds of like genotype. Curve R, disease progress curve for a completely random mixture of 25% C649 and 75% X421. Each point is the mean of three replications.

genotype units, and the completely random mixture was 1,414, 785, and 569 pustules-days, respectively. The probability level for the difference in the ADPC between the completely random mixture and the mixture of large genotype units was 0.44, whereas the probability level for the difference between the pure-line check and the mixture of large genotype units was 0.04.

With general epidemics, the completely random mixture decreased epidemic development more effectively than the mixture of large genotype units (Fig. 3). In fact, the disease progress curve for the mixture of large genotype units was nearly identical to that of the pure-line susceptible check. The ADPC for the pure-line check, the mixture of large genotype units, and the completely random mixture was 2,772, 2,580, and 853 pustules-days, respectively. The probability level for the difference in the ADPC between the two mixture treatments was <0.0001. The probability level for the difference in the ADPC between the pure-line check and the mixture of large genotype units was 0.49.

For all oat populations, epidemics increased less rapidly with a focal than with a general distribution of initial inoculum. The ADPC for the focally inoculated, pure-line susceptible plots was 49% less than the ADPC for the generally inoculated, pure-line susceptible plots; the probability level for this difference was 0.0005.

DISCUSSION

In 1984, disease developed less rapidly in focally inoculated than in generally inoculated plots. Similarly, Schmitt et al (20) found that wheat stem rust (induced by *Puccinia graminis* f. sp. *tritici* Erickss. and Henn.) did not increase as rapidly when initial inoculum was concentrated in a single focus at the center of a 122-m diameter plot than when the same amount of initial inoculum was dispersed in 30 foci within a 6.1-m diameter area in the center of the same-sized plot. By using a computerized simulation model, Kampmeijer and Zadoks (12) found that simulated epidemics did not increase as rapidly when initial inoculum was placed in a single focus than when the same amount of initial inoculum was dispersed in 400 foci placed uniformly over the host population. There was very little difference, however, between epidemics initiated with 16 versus 400 initial foci. Our results indicate that increasing host genotype unit area reduces the efficacy of host mixtures for oat crown rust control in a general epidemic. Results from epidemics initiated from an external source of inoculum in 1983 were similar to those from the artificial general inoculation in 1984. In 1984, there was a smaller effect of host genotype unit area on the efficacy of the mixture when initial disease was concentrated in a single focus. This is similar to results reported for focal epidemics of oat crown rust in Iowa (15). Computerized simulation studies also indicate that, with small grain rusts, increasing genotype unit area reduces the efficacy of host mixtures more with general than with focal epidemics (*unpublished*).

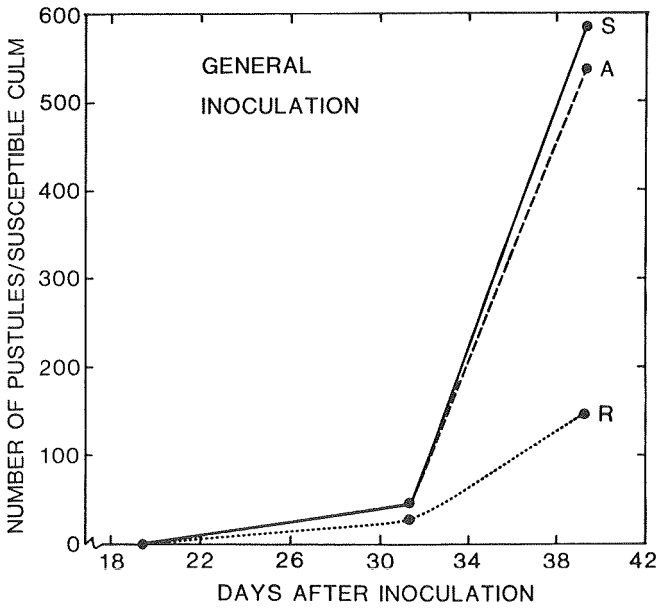

Fig. 3. Oat crown rust epidemics initiated in 1984 with the same amount of initial inoculum as in Fig. 2, but the inoculum was distributed uniformly over the plots. Number of oat crown rust pustules on susceptible (649) oat culms in field plots. Curve S, disease progress curve for a pure stand of C649. Curve A, disease progress curve for a mixture of 25% C649 and 75% X421 (immune) in which seeds were aggregated into blocks of 200 seeds of like genotype. Curve R, disease progress curve for a completely random mixture of 25% C649 and 75% X421. Each point is the mean of three replications.

Our results may explain why Barrett and Wolfe (1) found evidence indicating that lower seeding rates, which result in increased tillering of barley, reduced the efficacy of a barley cultivar mixture for the control of powdery mildew. Their study was conducted in Great Britain where barley powdery mildew infection is relatively heavy in most years (13,14) and, therefore, the initial disease was probably distributed generally. Our results are also consistent with those of Burdon and Chilvers (6) who found that changing the size of clusters of cress (*Lepidium sativum* L.) plants and adjacent areas of fallow soil had no effect on the rate of linear advance of damping-off (induced by *Pythium irregulare* Buisman) when epidemics were initiated from single foci. However, there was some association of increased cluster size with increased rate of disease progression when inoculum of *P. irregulare* was randomly dispersed in the soil.

At least two mechanisms may affect epidemic progression in a host mixture when genotype unit area is increased. First, as the area of genotype units is increased, disease is likely to increase more rapidly within susceptible genotype units. This is because the perimeter:area ratio decreases with increasing genotype unit area, resulting in a larger proportion of inoculum being retained within genotype units. Epidemic progression has been shown to increase with increasing area of experimental field plots (2,9,17). For example, the ADPC for potato late blight epidemics nearly doubled as plot size was increased from 0.81 to 29.16 m^2 (17). Second, if genotype unit area increases, and if the total area occupied by the genotype is constant, then the distances between susceptible genotype units will increase. Thus, the probability of inoculum movement from infected to noninfected genotype units will be decreased and the rate of colonization of susceptible genotype units will be reduced.

With general epidemics, most or all genotype units may be infected early in the epidemic. In that case, the rate of disease increase within genotype units should be a more important factor affecting the rate of epidemic progression than the distance between susceptible genotype units. Consequently, increasing the distance between units by making them larger would not have much retarding effect on the epidemic. With focal epidemics, however, the pathogen must disperse from the focus to previously noninfected genotype units. With focal oat crown rust epidemics, the retardation of movement of the pathogen among compatible genotype units may have been great enough to largely offset the increased rate of disease development within the larger genotype units. Burdon and Chilvers (6) used similar reasoning to explain their results with *P. irregulare*.

Our results suggest that the effect of genotype unit area on the effectiveness of host mixtures for disease control depends on the spatial distribution of initial disease. Therefore, disease control gained from diversification may be more variable and difficult to predict when host genotype unit area is large, e.g., with mixtures of crops with large plants, intercropping, or interfield diversification.

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